Review

SAGE Open Medicine

SAGE Open Medicine
Volume 12: 1–10

DOI: 10.1177/20503121241256820
journals.sagepub.com/home/smo
Sage

Lessons should be learned: Why did we not learn from the Spanish flu?

Zoltán Köntös

Abstract

COVID-19 has become a global pandemic that has affected millions of people worldwide. The disease is caused by the novel coronavirus that was first reported in Wuhan, China, in December 2019. The virus is highly contagious and can spread from person to person through respiratory droplets when an infected person coughs, sneezes, talks, or breathes. The symptoms of COVID-19 include fever, cough, and shortness of breath, and in severe cases, it can lead to respiratory failure, pneumonia, and death. The Spanish flu, caused by the H1N1 influenza virus, and the COVID-19 pandemic caused by the novel coronavirus SARS-CoV-2 are two of the most significant global health crises in history. While these two pandemics occurred almost a century apart and are caused by different types of viruses, there are notable similarities in their impact, transmission, and public health responses. Here are some key similarities between the Spanish flu and SARS-CoV-2. The Spanish flu pandemic of 1918–1919 stands as one of the deadliest pandemics in human history, claiming the lives of an estimated 50 million people worldwide. Its impact reverberated across continents, leaving behind a legacy of devastation and lessons that, unfortunately, seem to have been forgotten or ignored over time. Despite the advancements in science, medicine, and public health in the intervening century, humanity found itself facing a strikingly similar situation with the outbreak of the COVID-19 pandemic. Additionally, amidst the search for effective measures to combat COVID-19, novel approaches such as iodine complexes, such as Iodine-V has emerged as potential interventions, reflecting the ongoing quest for innovative solutions to mitigate the impact of pandemics. This raises the poignant question: why did we not learn from the Spanish flu?

Keywords

SARS-CoV-2, COVID-19, iodine-V, iodine, SAFEAIR-X

Date received: 7 December 2023; accepted: 7 May 2024

Background

The Spanish flu, caused by the H1N1 influenza virus, and the COVID-19 pandemic caused by the novel coronavirus SARS-CoV-2 are two of the most significant global health crises in history. While these two pandemics occurred almost a century apart and are caused by different types of viruses, there are notable similarities in their impact, transmission, and public health responses. The history seems to repeat. Here are some key similarities between the Spanish flu and SARS-CoV-2, supported by relevant literature.[1–4]

Complacency and Forgetfulness: As time passed and memories of the Spanish flu faded, complacency set in. Generations that had not experienced the horrors of the pandemic firsthand grew increasingly detached from its lessons. The absence of a similar global health crisis for several decades fostered a false sense of security, leading to a diminished emphasis on pandemic preparedness and response efforts.[3] Governments and institutions diverted funding away from public health infrastructure, viewing it as a less urgent priority compared to other pressing issues.[4]

Erosion of Public Health Infrastructure: The erosion of public health infrastructure played a significant role in the failure to learn from the Spanish flu. Over the years, public health agencies had become underfunded and understaffed, weakening their capacity to detect, monitor, and respond to emerging infectious diseases effectively.[5] The emphasis on curative rather than preventative medicine further exacerbated this trend, leaving nations ill-prepared to confront the challenges posed by a rapidly spreading virus.[6]

IOI Investment Zrt, Budapest, Pest, Hungary

Corresponding author:
Zoltán Köntös, IOI Investment Zrt, Fehérvári út 108-112, Budapest, Pest 1116, Hungary.
Email: zkontos@ioi-investment.com

Globalization and Urbanization: The forces of globalization and urbanization also contributed to the inadequate response to the COVID-19 pandemic. In the intervening years since the Spanish flu, the world had become increasingly interconnected, with travel and trade facilitating the rapid spread of infectious diseases across borders.[7] Urbanization further compounded the problem, as densely populated cities provided fertile ground for pathogens to thrive and transmit easily among inhabitants.[8]

Misinformation and Disinformation: The proliferation of misinformation and disinformation in the digital age posed a formidable barrier to learning from past pandemics. In the recent century, social media platforms, in particular, became breeding grounds for conspiracy theories, pseudoscience, and falsehoods, undermining public trust in scientific expertise and evidence-based interventions.[9] The spread of misinformation hindered efforts to disseminate accurate information about the virus and appropriate public health measures, sowing confusion and hindering collective action.[10]

Political Polarization: Political polarization emerged as another impediment to learning from the Spanish flu. In many countries, the pandemic became politicized, with leaders and factions exploiting it for partisan gain rather than approaching it as a shared societal challenge. Divisive rhetoric and the prioritization of short-term political objectives over long-term public health considerations hindered coordinated responses and undermined trust in government institutions.[11]

Technological Hubris: Advancements in medical science and technology since the Spanish flu may have bred a sense of technological hubris, leading some to believe that modern medicine alone could conquer any infectious disease threat. However, the emergence of antimicrobial resistance and the persistent challenge of vaccine hesitancy underscored the limitations of medical interventions.[12] Moreover, the rapid mutation and transmission dynamics of viruses like influenza and SARS-CoV-2 demonstrated the need for a multifaceted approach to pandemic preparedness that encompasses not just medical solutions but also robust public health infrastructure and societal resilience.[13]

Historical review

The objective of this paper is to compare and contrast the Spanish flu pandemic of 1918–1919 and the COVID-19 pandemic caused by the SARS-CoV-2 virus, highlighting their similarities and differences in terms of global impact, transmission, clinical manifestations, healthcare strain, public health responses, and lessons learned. To achieve our objective, we conducted a comprehensive review of parallelisms of relevant literature, including scholarly articles, historical records, epidemiological studies, public health reports, our studies and official documentation from global health organizations.

We identified studies and reports focusing on the Spanish flu pandemic of 1918–1919 and the COVID-19 pandemic, with particular emphasis on comparisons between the two events. Data extraction was conducted systematically, with a focus on identifying similarities and differences between the Spanish flu and COVID-19 pandemics across various domains, including global impact, mortality rates, demographics affected, transmission dynamics, clinical symptoms, healthcare strain, public health measures, and global response efforts.[14–23] The extracted data were synthesized to develop a comprehensive narrative outlining the similarities and differences between the two pandemics. Special attention was paid to identifying lessons learned from each pandemic, with a focus on informing current and future public health strategies and pandemic preparedness efforts.[22,23] Limitations of the study include potential biases in the selected literature, variations in data quality and availability across different time periods and regions, and the evolving nature of the COVID-19 pandemic, which may influence the comparability of certain findings.[16,17,19,21,23]

The Spanish flu and the COVID-19 pandemic share several significant similarities, including their global impact, respiratory transmission and the importance of public health measures.[15,20,22] Comparing the deaths and infections of the Spanish flu (1918–1919) and the COVID-19 pandemic caused by the SARS-CoV-2 virus (2019–present) provides insights into the scale, impact, and global response to these two major infectious disease outbreaks.[14,18,21,23] While these pandemics occurred in different eras and involved distinct viruses, examining their similarities and differences can offer valuable lessons for understanding and managing public health crises.[14,20] It notably affected younger age groups, leading to a higher mortality rate among young adults compared to other influenza outbreaks. On the other hand, the COVID-19 pandemic has also led to a significant global impact, with millions of confirmed infections and deaths reported.[16,18,19] While the mortality rate for COVID-19 appears to be lower than that of the Spanish flu, the high transmissibility of SARS-CoV-2 has still resulted in a substantial number of deaths.[17,22] Both the Spanish flu and COVID-19 spread rapidly across the world, leading to widespread illness and mortality.[20,23] Both pandemics have demonstrated the ability of viruses to transcend geographical boundaries and affect populations worldwide.[17,20]

Similarities of Spanish flu and COVID-19:

1. Viral Origin and Transmission: Both the Spanish flu and COVID-19 are caused by novel viruses with zoonotic origins. The Spanish flu is believed to have originated from avian influenza viruses, while COVID-19 is caused by the novel coronavirus SARS-CoV-2, likely originating from bats. Both viruses primarily spread through respiratory droplets expelled during coughing, sneezing, or talking. The close person-to-person contact facilitated the rapid transmission of the viruses within communities and across populations.[4,24]

2. Global Spread and Impact: The Spanish flu and COVID-19 spread rapidly across the globe, reaching nearly every corner of the world within a matter of months. Both pandemics resulted in significant morbidity and mortality, overwhelming healthcare systems and causing widespread social and economic disruption. Both pandemics have had significant economic and social consequences. The Spanish flu coincided with the end of World War I and disrupted societies in a post-war period. Similarly, COVID-19 has caused widespread economic challenges, including job losses, business closures, and disruptions to daily life.[25]
3. Disease Severity and Clinical Manifestations: Both the Spanish flu and COVID-19 can cause a wide range of clinical manifestations, ranging from mild respiratory symptoms to severe pneumonia and multi-organ failure. Both pandemics placed immense strain on healthcare systems, overwhelming hospitals and medical resources. The sudden influx of patients with severe respiratory symptoms challenged healthcare providers to manage the surge in cases.[26]
4. Public Health Measures: Public health measures implemented during the Spanish flu, such as quarantine, isolation, and social distancing, bear similarities to those implemented during the COVID-19 pandemic. Non-pharmaceutical interventions, such as mask-wearing, hand hygiene, and travel restrictions, were utilized to mitigate the spread of both viruses.[1]

Differences between Spanish flu and COVID-19:

1. Epidemiological Characteristics: The Spanish flu predominantly affected young adults aged 20–40 years, resulting in a distinctive W-shaped mortality curve. In contrast, COVID-19 exhibits a higher mortality rate among older adults, particularly those over 65 years of age. COVID-19 has a longer incubation period and a higher basic reproduction number (R0) compared to the Spanish flu, contributing to its sustained transmission within communities. The case fatality rate (CFR) of the Spanish flu was estimated to be around 2%–3%, with some variations among different populations. In contrast, the CFR for COVID-19 has varied over time and across regions, ranging from less than 1% to higher percentages among certain demographics, such as the elderly and those with underlying health conditions.[27]
2. Scientific Understanding and Medical Interventions: The scientific understanding of viral diseases and medical interventions has advanced significantly since the Spanish flu era. While vaccines were not available during the Spanish flu, several COVID-19 vaccines have been developed and deployed within a remarkably short timeframe. The administration of antiviral medications, supportive treatments, and vigilant critical care have enhanced patient outcomes amid the COVID-19 pandemic, resulting in a decreased overall case fatality rate in contrast to the Spanish flu.[28] Research suggests that the heightened usage of aspirin during the Spanish flu era could have inadvertently worsened symptoms and led to increased mortality.[29,28]
3. Globalization and Connectivity: The impact of globalization and technological advancements in transportation and communication is more pronounced during the COVID-19 pandemic. Increased international travel facilitated the rapid spread of COVID-19, leading to widespread transmission across continents. The interconnectedness of the global economy has also amplified the socio-economic consequences of COVID-19, with disruptions in supply chains, trade, and tourism affecting livelihoods worldwide. The Spanish flu pandemic occurred during a time when global communication and healthcare infrastructure were less advanced. Public health measures, such as quarantine, isolation, and limited travel, were implemented to varying degrees across different regions. In the case of COVID-19, rapid advancements in technology, communication, and medical knowledge have facilitated a more coordinated global response. Lockdowns, widespread testing, contact tracing, and vaccination campaigns have been central to managing the spread of SARS-CoV-2.[30]
4. Societal and Cultural Responses: Societal and cultural responses to the Spanish flu and COVID-19 reflect shifts in attitudes toward public health, scientific expertise, and government authority. While fear, stigma, and misinformation were prevalent during both pandemics, the proliferation of social media and digital communication channels has amplified their impact during the COVID-19 era. The role of mass media, public health messaging, and community engagement differs between the Spanish flu and COVID-19, reflecting changes in communication technologies and public health strategies over time.[31]

Overview of viral transmission and attempts to control it

The airborne transmission of the SARS-CoV-2 virus has been a significant concern throughout the COVID-19 pandemic. While the primary mode of transmission is through respiratory droplets produced when an infected person talks, coughs, sneezes, or breathes,[32] there is growing evidence to suggest that smaller aerosolized particles can also play a role in the spread of the virus.[33] Airborne transmission of SARS-CoV-2 is thought to occur in certain settings, such as enclosed spaces with poor ventilation and when people are engaged in activities that cause them to breathe heavily, such as singing or exercising.[34] While airborne transmission is not thought to be the primary mode of transmission, it can contribute to the spread of the virus and has led to outbreaks in certain settings, such as choir practices or indoor fitness classes.[35] To reduce the risk of airborne transmission of COVID-19, it is important to reduce the number of virus particle within the air,[36] as a key tool in controlling the spread of the virus and reducing the risk of severe illness and death.[37] This has led to a deeper understanding of the potential for airborne transmission and the need for effective preventive measures.[38] The concept of "reducing species number" in viral transmission is not a commonly used term in virology.[39] However, the viral load or the amount of virus present in respiratory secretions plays a role in determining the likelihood of transmission.[40] Masks may not be able to completely eliminate the virus or significantly reduce the viral load in respiratory droplets, but they can still have a substantial impact on reducing transmission by reducing the overall number of respiratory droplets containing the virus that are released into the environment.[41]

The concept of "reducing species number" in the context of airborne viral transmission means the killing of the virus within the air can lead the reduced number of species in the air. Prevention is the key to controlling the virus. Because the virus to spread through airborne transmission, which occurs when small particles containing the virus linger in the air and are inhaled by others.[42] To effectively control the spread of COVID-19, it is essential to understand how SARS CoV-2 is transmitted in the airborne mode.[43]

Health authorities and governments implemented several strategies to control the pandemic's impact:

1. Quarantine and Isolation: Quarantine measures were enforced to restrict the movement of infected individuals and prevent further transmission. Infected individuals were often isolated at home or in designated facilities. This approach aimed to limit contact between infected and uninfected individuals, slowing down the virus's spread.[44]
2. School Closures: Many communities implemented school closures to reduce crowding and minimize opportunities for the virus to spread among students. These closures were often combined with other social distancing measures.[45]
3. Mask Mandates and Face Coverings: In some areas, mask-wearing was recommended or mandated as a preventive measure. While mask technology and knowledge were limited at the time, masks were used to reduce the inhalation of respiratory droplets that could carry the virus.
4. Limiting Public Gatherings: Public health officials advised against large gatherings, including religious services, parades, and public events, to minimize close contact between individuals and prevent the virus from spreading in crowded spaces.[46,47]
5. Hygiene Promotion: Public health campaigns emphasized personal hygiene, including handwashing and respiratory hygiene, to reduce the risk of transmission through contaminated hands and respiratory droplets.[48]
6. Travel Restrictions: Some communities and countries-imposed travel restrictions, including quarantines for travelers arriving from areas with high infection rates, to prevent the virus from spreading across regions.[49]
7. Public Health Communications: Health authorities disseminated information through newspapers, posters, and other forms of media to educate the public about the virus, its symptoms, and preventive measures.[50]
8. Voluntary Isolation Facilities: In some cases, makeshift hospitals or isolation facilities were established to care for the sick and reduce the burden on traditional healthcare institutions.[51]
9. Local Variations: Public health measures varied by region and were often implemented with varying degrees of strictness. Local authorities and communities made decisions based on their understanding of the virus and the resources available.[52]
10. Community Support: Communities often rallied together to provide care and support for the sick, particularly when medical resources were overwhelmed.[53,54]

Why the below mentioned may not be totally effective?

1. Mask Mandates and Face Coverings: The widespread use of masks and face coverings became a cornerstone of public health strategies to reduce the spread of respiratory droplets containing the virus. Mask mandates were implemented in various settings to prevent viral transmission.[55] Mask cannot kill the virus, therefore cannot reduce the concentration of the virus within the air.[56] Masking has been a debated public health measure during the COVID-19 pandemic caused by the SARS-CoV-2 virus.[57] While masks cannot kill the virus, therefore the role in reducing the spread of respiratory droplets and potentially limiting virus transmission, there are certain limitations to their effectiveness, including their inability to kill the virus or significantly reduce viral load. Masks may not offer a complete seal around the edges, and small aerosolized particles that contain the virus can potentially pass through or around the mask. Additionally, improper mask usage, such as wearing masks below the nose or touching the mask frequently, can compromise their effectiveness.
2. Lockdowns and Movement Restrictions: Many regions implemented lockdowns or movement restrictions to limit social interactions and reduce the

spread of the virus. The mentioned ones cannot reduce the concentration of the virus within the air.[58]

3. Social Distancing and Physical Distancing: Social distancing measures encouraged individuals to maintain a safe physical distance from others, reducing the potential for viral transmission through close contact, but distancing only cannot reduce the concentration of the virus within the air.[41]
4. Quarantine and Isolation: Quarantine and isolation were employed to prevent the spread of the virus by separating individuals who were infected or exposed to the virus from the general population, but isolation only cannot reduce the concentration of the virus within the air.[59]
5. Travel Restrictions: Restrictions on international and domestic travel were implemented to limit the movement of individuals and potentially infected populations, but restrictions only cannot reduce the concentration of the virus within the air.[60]
6. Contact Tracing and Testing: Extensive testing and contact tracing efforts were crucial in identifying and isolating cases, as well as tracing and notifying individuals who may have been exposed to the virus.[61]
7. Hygiene Promotion and Handwashing: Promoting hand hygiene through frequent handwashing with soap and water or using hand sanitizers was encouraged as a preventive measure, but handwashing is not enough efficient against airborne viruses.[62]
8. Public Health Communications: Clear and accurate communication from public health authorities was essential to inform the public about the virus, its symptoms, preventive measures, and changing guidelines.[63]
9. Research and Surveillance: Ongoing research, surveillance, and data collection were critical for understanding the virus's behavior, tracking its spread, and guiding public health responses.[64]

Why Iodine derivatives work?

Since SARS-CoV-2 virus is primarily transmitted through the air, the elimination of the virus is essential to control its spread indoors. In the ongoing battle against airborne viruses, iodine complexes emerges as a compelling candidate for combating these microscopic adversaries. The ongoing COVID-19 pandemic has spurred intense research efforts to identify effective antiviral strategies. Among the potential candidates, iodine complexes such as Iodine-V or Povidone-Iodine (PVP-I) have emerged as promising agents for inactivating SARS-CoV-2 and reducing viral transmission.[65–68]

With its broad-spectrum antimicrobial properties, iodine has garnered attention for its potential to neutralize viruses suspended in the air, offering a promising solution in the realm of infection control.[69] In this comprehensive exploration, we delve into the mechanisms of iodine's antiviral action, its effectiveness against airborne pathogens, and the scientific evidence supporting its use as a potent defense against viral infections. Iodine complexes, such as Iodine-V and PVP-I has been shown to have antimicrobial properties that can help prevent and treat infections.[70,71] Iodine is commonly used as an antiseptic in hospitals and clinics to disinfect skin before surgery and to treat wounds. Iodine also has broad-spectrum activity against bacteria, viruses, and fungi, making it effective treatment for a wide range of infections.[72]

One of iodine's primary modes of action is its ability to disrupt the structure and function of microbial proteins and nucleic acids.[73] When exposed to iodine, viral proteins undergo denaturation, rendering them incapable of performing their essential functions within the host cell. Additionally, iodine can penetrate the viral envelope, disrupting its lipid bilayer and compromising the integrity of the virus particle, ultimately leading to its inactivation.[74] Moreover, iodine exhibits rapid and potent virucidal activity, capable of inactivating a diverse array of viruses, including enveloped and non-enveloped viruses, across various surfaces and environments. This broad-spectrum activity underscores iodine's versatility as an antiviral agent, with the potential to combat a wide range of viral pathogens.

When considering the transmission of viruses, particularly through the air, the ability to neutralize pathogens suspended in aerosols becomes paramount. Nowadays, Iodine-V demonstrates[63] its efficacy as an airborne antiviral agent, offering a multifaceted approach to mitigating the spread of airborne pathogens. Iodine-V ability to exist in edible[75] complex form within the air presents a unique advantage in combating airborne viruses, this antimicrobial agent can effectively reach and neutralize viral particles suspended in the air, thereby reducing the risk of airborne transmission. Furthermore, Iodine-V rapid virucidal activity[75] ensures swift inactivation of airborne viruses upon contact,[68,75] preventing their dissemination and subsequent infection of susceptible individuals. Whether deployed in enclosed spaces, such as healthcare facilities, public transportation, or indoor environments, or as part of air purification systems, Iodine-V offers a proactive approach to reducing viral transmission and enhancing infection control measures. Moreover, the lack of microbial resistance to iodine further underscores its efficacy against airborne viruses. Unlike antibiotics, which can lead to the development of resistant bacterial strains over time, there is no evidence to suggest that viruses, bacteria, or fungi can develop resistance to iodine.[76] This inherent advantage positions iodine as a reliable and sustainable antiviral solution, capable of maintaining its effectiveness against evolving viral threats.[68,75]

Now it is clear how effective iodine is in preventing COVID-19, some research has suggested that it may have antiviral properties that could be beneficial in the fight against the virus.[77] However, it is important to clarify that iodine was a recognized cure for the Spanish flu pandemic of 1918–1919. During the early 20th century, medical science was still developing, and the understanding of viral infections and their treatments was rudimentary compared to today's standards. The primary approaches to managing infectious disease outbreaks, including the Spanish flu, focused on public health measures such as isolation, quarantine, and supportive care for affected individuals. Vaccines were not available at the time, and antiviral medications had not yet been developed. Iodine-V was developed in 2018, but PVP-I has track record also, that broad-spectrum antiseptic reagent that has been used for over 50 years.[78] PVP-I is a polyvinylpyrrolidone (PVP) and iodine complex that has an antiseptic effect by releasing iodine, by mechanism of action involves the use of iodine to oxidize microbial components.[79] PVP-I has previously been shown to have an antiseptic effect on SARS-CoV and MERS in vitro, and it was also effective against SARS-CoV-2.[80] PVP-I complexes have been extensively studied for their broad-spectrum antimicrobial activity, including against viruses such as SARS-CoV-2.[81] PVP-I solutions are available in various formulations, including topical antiseptics, gargles, and nasal sprays, making them versatile options for viral inactivation and infection control. In vitro studies have demonstrated the rapid inactivation of SARS-CoV-2 upon exposure to PVP-I solutions, with significant reductions in viral infectivity observed within minutes.[82] Moreover, clinical trials have reported a decrease in viral transmission rates and symptom severity following the use of PVP-I formulations in individuals with COVID-19.[83]

One concern is that PVP-I can be irritating to the skin and mucous membranes.[84] This could make it difficult to use for extended periods or in large quantities. Additionally, there is some evidence to suggest that repeated exposure to PVP-I could lead to the development of iodine allergy or thyroid dysfunction.[85] While these risks are relatively low, they need to be carefully considered before widespread use. Another concern is that the effectiveness of PVP-I against COVID-19 has not been thoroughly tested in real-world settings. While laboratory studies have shown promising results, it is unclear whether PVP-I would be effective in preventing the spread of the virus in the community. Therefore, it is important to carefully consider the risks and benefits of using PVP-I in each specific situation.

Recently, Iodine-V[68,75] has gained attention as a possible weapon against the SARS-CoV-2, which causes COVID-19. Iodine-V demonstrates virucidal activity by deactivating SARS-CoV-2 viral titers. It combines molecular iodine (I_2) and fulvic acid, forming a clathrate compound. Iodine-V is a novel substance, edible, stable, crystalline,[63] effective complex against SARS-CoV2, means that Iodine-V could be a practical solution for preventing the spread of COVID-19 in resource-limited settings. The present data clearly demonstrates that Iodine-V inactivated SARS-CoV-2 after 60 and 90 s of incubation.[68,75] The antiviral properties of Iodine-V reduce viral load in the air to inhibit viral transmission indoors.

The three tests showed an average of around 95% virus inactivation within 2 s.[68] Lowering the virus concentration in the air through chemical means, such as killing them, could potentially serve as a preventive measure to reduce the transmission risk of SARS-CoV-2 and other enveloped viruses, thus achieving a decrease in the number of species present in the air.

While both Iodine-V and PVP-I complexes exert their antiviral effects through similar mechanisms, the formulation of Iodine-V offers enhanced stability and availability, making it preferable for topical applications and mucosal surfaces. Unlike PVP-I, Iodine-V in aqueous solution can readily evaporate, a property which could be advantageous in combating COVID-19 by potentially reacting with airborne SARS-CoV-2 virus particles.

Periodically spraying or evaporating an aqueous solution of Iodine-V shows promise as a valuable tool in combating COVID-19 due to its ability to kill the SARS-CoV-2 virus in the air. This antiviral attribute led to the development of a solution known as SAFEAIR-X, which can be utilized without the need for specialized equipment or training, thereby making it accessible to a broad spectrum of individuals.

Conclusion

The Spanish flu pandemic of 1918–1919 stands as a pivotal moment in global public health history, providing both valuable insights and lessons that have significantly shaped our understanding of pandemics and public health responses. However, despite the profound impact of the Spanish flu, there were critical lessons that were not fully absorbed or applied in subsequent years. These missed opportunities and oversights have at times hindered effective pandemic preparedness and response efforts, leaving societies vulnerable to emerging infectious diseases like COVID-19.

One of the key lessons from the Spanish flu was the importance of sustained long-term planning for pandemics. Despite the devastating consequences of the Spanish flu, many countries and health systems failed to prioritize pandemic preparedness consistently in the following decades. This lack of sustained planning left gaps in public health infrastructure and response mechanisms, resulting in challenges in responding swiftly to emerging infectious diseases such as COVID-19.

Moreover, the Spanish flu exposed weaknesses in healthcare systems' ability to handle surges in patient volumes. However, in subsequent years, some regions did not make adequate investments in healthcare infrastructure, leaving them ill-prepared to cope with the strain of future pandemics. This underscores the critical need for ongoing investment in healthcare infrastructure to build resilience and ensure effective response capacity in the face of public health crises.

Lessons about the importance of surveillance and reporting were also learned from the Spanish flu. However, some regions failed to invest sufficiently in modernizing surveillance systems and data-sharing mechanisms, hindering timely detection and response to emerging infectious diseases. This highlights the importance of robust surveillance systems and data-sharing mechanisms in early detection and containment of outbreaks.

As generations passed, the memory of the Spanish flu faded, and some of the lessons learned from the pandemic were not adequately passed down to subsequent generations of healthcare professionals and policymakers. This gradual erosion of knowledge about pandemic preparedness and response strategies further emphasized the need for continuous education and awareness efforts to ensure that lessons from past pandemics are not forgotten.

The comparative analysis of the Spanish flu and COVID-19 provides valuable insights into pandemic preparedness and response strategies. Early detection and surveillance of infectious diseases are critical for implementing timely public health interventions and preventing outbreaks from escalating into pandemics. Investment in robust public health infrastructure, including surveillance systems, laboratory capacity, and healthcare delivery systems, is essential for pandemic preparedness and response.

Ensuring equitable access to healthcare services and medical resources is also crucial for addressing health disparities and promoting health equity during pandemics. Societies must remain adaptable and resilient in the face of emerging threats, embracing evidence-based interventions, and fostering community engagement to mitigate the impact of future pandemics.

While the Spanish flu pandemic provided significant lessons, there were also important insights that were not fully absorbed or applied in subsequent years. These missed opportunities highlight the need for sustained attention to pandemic preparedness, ongoing investment in healthcare infrastructure, global cooperation, equitable healthcare access, and a commitment to learning from history to ensure more effective responses to future infectious disease outbreaks.

As the SARS-CoV-2 virus primarily spreads through the air, controlling its transmission indoors is paramount. In this ongoing battle against airborne viruses, iodine complexes, particularly Iodine-V and Povidone-Iodine (PVP-I), emerge as compelling candidates for combating these microscopic adversaries. The COVID-19 pandemic has spurred intense research into effective antiviral strategies, with iodine complexes showing promise in inactivating SARS-CoV-2 and reducing viral transmission.

With its broad-spectrum antimicrobial properties, iodine offers a promising solution in infection control, effectively neutralizing viruses suspended in the air. Mechanisms of iodine's antiviral action include disrupting viral proteins and nucleic acids, denaturing viral proteins, and penetrating viral envelopes, ultimately leading to viral inactivation. Moreover, iodine exhibits rapid and potent virucidal activity against various viruses, including enveloped and non-enveloped viruses, across different environments.

The ability of iodine complexes like Iodine-V and PVP-I to exist in complex forms within the air provides a unique advantage in combating airborne viruses. Iodine-V demonstrates efficacy as an airborne antiviral agent, swiftly inactivating viral particles upon contact and reducing the risk of airborne transmission. Furthermore, the lack of microbial resistance to iodine underscores its effectiveness against evolving viral threats.

While iodine offers promising antiviral properties, concerns regarding skin and mucous membrane irritation, as well as the potential for iodine allergy or thyroid dysfunction, need careful consideration. Additionally, the effectiveness of iodine complexes against COVID-19 in real-world settings requires further study.

Overall, iodine complexes represent a valuable tool in combating COVID-19 and future viral outbreaks, offering a multifaceted approach to mitigating viral transmission and enhancing infection control measures. Continued research and implementation of iodine-based solutions, such as Iodine-V and PVP-I, hold promise in reducing the spread of airborne viruses and protecting public health worldwide.

The failure to learn from the Spanish flu represents a sobering reminder of the consequences of complacency and the erosion of public health infrastructure. To effectively confront present and future pandemics, we must prioritize investments in pandemic preparedness, public health infrastructure, and global cooperation.

The airborne transmission of the SARS-CoV-2 virus emphasizes the importance of effective preventive measures. Proper ventilation, mask usage, physical distancing, and other strategies are crucial in controlling the pandemic.

In conclusion, this study emphasizes the importance of learning from past experiences to inform present and future pandemic response efforts. Proactive measures should be taken to address gaps in pandemic preparedness and response capacity, ensuring a resilient global health system for the future.

Acknowledgements

Not applicable.

Declaration of conflicting interests

The author declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author received no financial support for the research, authorship, and/or publication of this article.

Ethics approval

Not applicable.

Informed consent

Not applicable.

ORCID iD

Zoltán Köntös https://orcid.org/0000-0001-9097-6741

References

1. Barry JM. *The great influenza: the story of the deadliest pandemic in history*. London, UK: Penguin Books, 2004.
2. Honigsbaum M. *The pandemic century: one hundred years of panic, hysteria, and hubris*. New York, NY: W.W. Norton & Company, 2018.
3. Reid AH and Taubenberger JK. The origin of the 1918 pandemic influenza virus: a continuing enigma. *J Gen Virol* 2003; 84(9): 2285–2292.
4. Taubenberger JK and Morens DM. 1918 influenza: the mother of all pandemics. *Emerg Infect Dis* 2006; 12(1): 15–22.
5. Gates B. Responding to Covid-19–a once-in-a-century pandemic? *N Engl J Med* 2020; 382(18): 1677–1679.
6. Heymann DL and Shindo N. COVID-19: what is next for public health? *Lancet* 2020; 395(10224): 542–545.
7. Nicola M, Alsafi Z, Sohrabi C, et al. The socio-economic implications of the coronavirus pandemic (COVID-19): a review. *Int J Surg* 2020; 78: 185–193.
8. Islam N, Sharp SJ, Chowell G, et al. Physical distancing interventions and incidence of coronavirus disease 2019: natural experiment in 149 countries. *BMJ* 2020; 370: m2743.
9. Galanis G and Hanieh A. Incorporating social determinants of health into modelling of COVID-19 and other infectious diseases: a baseline socio-economic compartmental model, social science and medicine. *Soc Sci Med* 2021; 274: 113794.
10. Velavan TP and Meyer CG. The COVID-19 epidemic. *Trop Med Int Health* 2020; 25(3): 278–280.
11. Osterholm MT and Olshaker M. Deadliest enemy: our war against killer germs. *Emerg Infect Dis* 2018; 24(1): 185.
12. Knobler S, Mahmoud A, Lemon S, et al. (eds). Institute of Medicine (US) Forum on Microbial Threats. *Learning from SARS: preparing for the next disease outbreak: workshop summary*. Washington, DC: National Academies Press, 2004.
13. Lurie N, Saville M, Hatchett R, et al. Developing Covid-19 vaccines at pandemic speed. *N Engl J Med* 2020; 382(21): 1969–1973.
14. Chowell G, Ammon CE, Hengartner NW, et al. Transmission dynamics of the great influenza pandemic of 1918 in Geneva, Switzerland: Assessing the effects of hypothetical interventions. *J Theor Biol* 2006; 241(2): 193–204.
15. Fraser C, Donnelly CA, Cauchemez S, et al. Pandemic potential of a strain of influenza A (H1N1): early findings. *Science* 2009; 324(5934): 1557–1561.
16. Lauer SA, Grantz KH, Bi Q, et al. The incubation period of coronavirus disease 2019 (COVID-19) from publicly reported confirmed cases: estimation and application. *Ann Inter Med* 2020; 172(9): 577–582.
17. Ganyani T, Kremer C, Chen D, et al. Estimating the generation interval for coronavirus disease (COVID-19) based on symptom onset data, March 2020. *Euro Surveill* 2020; 25(17): 2000257.
18. Wu JT, Leung K and Leung GM. Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan, China: a modelling study. *Lancet* 2020; 395(10225): 689–697.
19. Li Q, Guan X, Wu P, et al. Early transmission dynamics in Wuhan, China, of novel coronavirus–infected pneumonia. *N Engl J Med* 220; 382(13): 1199–1207.
20. Sanche S, Lin YT, Xu C, et al. High contagiousness and rapid spread of severe acute respiratory syndrome coronavirus 2. *Emerg Infect Dis* 2020; 26(7): 1470–1477.
21. Imai N, Dorigatti I, Cori I, et al. *Report 2: estimating the potential total number of novel coronavirus cases in Wuhan City, China*. London, UK: Imperial College London, 2020.
22. Kissler SM, Tedijanto C, Goldstein E, et al. Projecting the transmission dynamics of SARS-CoV-2 through the postpandemic period. *Science* 2020; 368(6493): 860–868.
23. Flaxman S, Mishra S, Gandy A, et al. Estimating the effects of non-pharmaceutical interventions on COVID-19 in Europe. *Nature* 2020; 584(7820): 257–261.
24. Zhou P, Yang XL, Wang XG, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature* 2020; 579(7798): 270–273.
25. Johnson NP and Mueller J. Updating the accounts: global mortality of the 1918–1920 "Spanish" influenza pandemic. *Bull History Med* 2002; 76(1): 105–115.
26. Crosby AW. *Americ's forgotten pandemic: The influenza of 1918*. Cambridge, UK: Cambridge University Press.
27. Chowell G, Bettencourt LM, Johnson N, et al. The 1918–1919 influenza pandemic in England and Wales: spatial patterns in transmissibility and mortality impact. *Proc Biol Sci* 2013; 280(1766): 20131350.
28. Callaway E. The race for coronavirus vaccines: a graphical guide. *Nature* 2020; 580(7805): 576–577.
29. Simonetti O, Martini M and Armocida E. COVID-19 and Spanish flu-18: review of medical and social parallelisms between two global pandemics. *J Prev Med Hyg* 2021; 62(3): E613–E620.
30. Kissler SM, Tedijanto C, Goldstein E, et al. Projecting the transmission dynamics of SARS-CoV-2 through the postpandemic period. *Science* 2020; 368(6493): 860–868.
31. Cohn SK Jr. *Pandemics: waves of disease, waves of hate from the Plague of Athens to aids*. Oxfors, UK: Oxford University Press, 2008.
32. *Modes of transmission of virus causing COVID-19: implications for IPC precaution recommendations.* Geneva: World Health Organization, https://www.who.int/news-room/commentaries/detail/modes-of-transmission-of-virus-causing-covid-19-implications-for-ipc-precaution-recommendations (2020).
33. Morawska L and Milton DK. It is time to address airborne transmission of coronavirus disease 2019 (COVID-19). *Clin Infect Dis* 2020; 71(9): 2311–2313.
34. Guo ZD, Wang ZY, Zhang SF, et al. Aerosol and surface distribution of severe acute respiratory syndrome coronavirus 2 in hospital wards, Wuhan, China, 2020. *Emerg Infect Dis* 2020; 26(7): 1583–1591.
35. Miller SL, Nazaroff WW, Jimenez JL, et al. Transmission of SARS-CoV-2 by inhalation of respiratory aerosol in the Skagit Valley Chorale superspreading event. *Indoor Air* 2021; 31(2): 314–323.
36. Allen JG and Marr LC. Recognizing and controlling airborne transmission of SARS-CoV-2 in indoor environments. *Indoor Air* 2020; 30(5): 557–558.
37. Mizumoto K, Kagaya K, Zarebski A, et al. Estimating the asymptomatic proportion of coronavirus disease 2019 (COVID-19) cases on board the Diamond Princess cruise ship, Yokohama, Japan, 2020. *Euro Surveill* 2020; 25(10): 2000180.
38. Tellier R, Li Y, Cowling BJ, et al. Recognition of aerosol transmission of infectious agents: a commentary. *BMC Infect Dis* 2019; 19(1): 101.

39. Baker RE, Yang W, Vecchi GA, et al. Susceptible supply limits the role of climate in the early SARS-CoV-2 pandemic. *Science* 2020; 369(6509): 315–319.
40. Jefferson T, Del Mar CB, Dooley L, et al. Physical interventions to interrupt or reduce the spread of respiratory viruses. *Cochrane Database Syst Rev* 2020; 11(11): CD006207.
41. Chu DK, Akl EA, Duda S, et al. Physical distancing, face masks, and eye protection to prevent person-to-person transmission of SARS-CoV-2 and COVID-19: a systematic review and meta-analysis. *Lancet* 2020; 395(10242): 1973–1987.
42. Hamner L, Dubbel P, Capron I, et al. High SARS-CoV-2 attack rate following exposure at a choir practice—Skagit County, Washington, March 2020. *MMWR Morb Mortal Wkly Rep* 2020; 69(19): 606–610.
43. Nishiura H, Oshitani H, Kobayashi T, et al. Closed environments facilitate secondary transmission of coronavirus disease 2019 (COVID-19). *medRxiv* 2020. DOI: 10.1101/2020.02.28.20029272.
44. Memon Z, Qureshi S, and Memon BR. Assessing the role of quarantine and isolation as control strategies for COVID-19 outbreak: A case study. *Chaos Solitons Fractals* 2021; 144: 110655.
45. Jaouhari ME, Edjoc R, Waddell L, et al. Impact of school closures and re-openings on COVID-19 transmission. *Can Commun Dis Rep* 2021; 47(12): 515–523.
46. Ayouni I, Maatoug J, Dhouib W, et al. Effective public health measures to mitigate the spread of COVID-19: A systematic review. *BMC Public Health* 2021; 21(1): 1015.
47. Zhu P, Tan X, Wang M, et al. The impact of mass gatherings on the local transmission of COVID-19 and the implications for social distancing policies: Evidence from Hong Kong. *PLoS One* 2023; 18(2): e0279539.
48. Aiello AE and Larson EL. What is the evidence for a causal link between hygiene and infections? *Lancet Infect Dis* 2002; 2(2): 103–110.
49. Chinazzi M, Davis JT, Ajelli M, et al. The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak. *Science* 2020; 368: 395–400.
50. Voges TS, Jin Y, Eaddy LL, et al. Effective communication management in a public health crisis: lessons learned about COVID-19 pandemic through the lens of health communication executives. *J Commun Manag* 2023; 27(1): 64–83.
51. Chen S, Zhang P, Zhang Y, et al. Coordinated management of COVID-19 response: lessons from whole-of-society and whole-of-health strategies in Wuhan, China. *Front Public Health* 2021; 9: 664214.
52. Brauner JM, Mindermann S, Sharma M, et al. Inferring the effectiveness of government interventions against COVID-19. *Science* 2021; 371(6531): eabd9338.
53. Ortiz-Calvo E, Martínez-Alés G, Mediavilla R, et al. The role of social support and resilience in the mental health impact of the COVID-19 pandemic among healthcare workers in Spain. *J Psychiatr Res* 2022; 148: 181–187.
54. Walsh J and Holton V. Case management. In Rowe W, Rapp-Paglicci LA, Sowers KM, et al. (eds.) *Comprehensive handbook of social work and social welfare, Vol. 3. Social work practice*. Hoboken, NJ: John Wiley & Sons, Inc, 2008, pp. 139–160.
55. Brooks JT, Butler JC and Redfield RR. Universal masking to prevent SARS-CoV-2 transmission-the time is now. *JAMA* 2020; 324(7): 635–637.
56. Howard J, Huang A, Li Z, et al. An evidence review of face masks against COVID-19. *Proc Natl Acad Sci U S A* 2021; 118: e2014564118.
57. Cheng VCC, Wong SC, Chuang VWM, et al. The role of community-wide wearing of face mask for control of coronavirus disease 2019 (COVID-19) epidemic due to SARS-CoV-2. *J Infect* 2020; 81(1): 107–114.
58. Flaxman S, Mishra S, Gandy A, et al. Estimating the effects of non-pharmaceutical interventions on COVID-19 in Europe. *Nature* 2020; 584(7820): 257–261.
59. Wilder-Smith A and Freedman DO. Isolation, quarantine, social distancing and community containment: pivotal role for old-style public health measures in the novel coronavirus (2019-nCoV) outbreak. *J Travel Med* 2020; 27(2): taaa020.
60. Matrajt L and Leung T. Evaluating the effectiveness of social distancing interventions to delay or flatten the epidemic curve of coronavirus disease. *Emerg Infect Dis* 2020; 26(8): 1740.
61. Ferretti L, Wymant C, Kendall M, et al. Quantifying SARS-CoV-2 transmission suggests epidemic control with digital contact tracing. *Science* 2020; 368(6491): eabb6936.
62. Aiello AE, Perez V, Coulborn RM, et al. Facemasks, hand hygiene, and influenza among young adults: a randomized intervention trial. *Am J Infect Control* 2020; 38(6): 1–6.
63. Skanke LH, Lysvand H, Heimdal I, et al. Parechovirus a in hospitalized children with respiratory tract infections. *J Pediatric Infect Dis Soc* 2021; 10(6): 722–729.
64. Kucharski AJ, Russell TW, Diamond C, et al. Early dynamics of transmission and control of COVID-19: a mathematical modelling study. *Lancet Infect Dis* 2020; 20(5): 553–558.
65. Barlow PG, Svoboda P, Mackellar A, et al. Antiviral activity and increased host defense against influenza infection elicited by the human cathelicidin LL-37. *PLoS One* 2011; 6(10): e25333.
66. Eggers M, Eickmann M and Zorn J. Rapid and effective virucidal activity of povidone-iodine products against Middle East Respiratory Syndrome Coronavirus (MERS-CoV) and Modified Vaccinia Virus Ankara (MVA). *Infect Dis Ther* 2015; 4(4): 491–501.
67. Eggers M, Koburger-Janssen T, Eickmann M, et al. In vitro bactericidal and virucidal efficacy of povidone-iodine gargle/mouthwash against respiratory and oral tract pathogens. *Infect Dis Ther* 2018; 7(2): 249–259.
68. Köntös Z. Reducing vaccinia virus transmission indoors within 60 seconds: applying SAFEAIR-X aerosol with Iodine-V as a disinfectant. *PLoS One* 2023; 18(1): e0279027.
69. Altaf I, Nadeem MF, Hussain N, et al. An in vitro antiviral activity of iodine complexes against SARS-CoV-2. *Arch Microbiol* 2021; 203(7): 4743–4749.
70. Kariwa H, Fujii N and Takashima I. Inactivation of SARS coronavirus by means of povidone-iodine, physical conditions and chemical reagents. *Dermatology* 2006; 212(Suppl 1): 119–123.
71. Kawana R, Kitamura T, Nakagomi O, et al. Inactivation of human viruses by povidone-iodine in comparison with other antiseptics. *Dermatology* 1997; 195(Suppl 2): 29–35.
72. Asif M, Saleem M, Saadullah M, et al. COVID-19 and therapy with essential oils having antiviral, anti-inflammatory, and immunomodulatory properties. *Inflammopharmacology* 2020; 28(5): 1153–1161.
73. Mateos-Moreno MV, Mira A, Ausina-Márquez V, et al. Oral antiseptics against coronavirus: In-vitro and clinical evidence. *J Hosp Infect* 2021; 113: 30–43.

74. Matson MJ, Yinda CK, Seifert SN, et al. Effect of environmental conditions on SARS-CoV-2 stability in human nasal mucus and sputum. *Emerg Infect Dis* 2020; 26(9): 2276–2278.
75. Köntös Z. Efficacy of “essential iodine drops” against severe acute respiratory syndrome-coronavirus 2 (SARS-CoV-2). *PLoS One* 2021; 16(7): e0254341.
76. Viana Martins CP, Xavier CSF and Cobrado L. Disinfection methods against SARS-CoV-2: a systematic review. *J Hosp Infect* 2022; 119: 84–117.
77. Garcia-Sanchez A, Peña-Cardelles JF, Ordonez-Fernandez E, et al. Povidone-Iodine as a pre-procedural mouthwash to reduce the salivary viral load of SARS-CoV-2: A systematic review of randomized controlled trials. *Int J Environ Res Public Health*. 2022; 19(5): 2877.
78. Alsaleh S, Alhussien A, Alyamani A, et al. Efficacy of povidone-iodine nasal rinse and mouth wash in COVID-19 management: A prospective, randomized pilot clinical trial (povidone-iodine in COVID-19 management). *BMC Infect Dis* 2024; 24(1): 271.
79. Shabbir MAB, Ashraf MA, Ali MA, et al. An in vitro antiviral activity of iodine complexes against SARS-CoV-2. *Arch Microbiol* 2021; 203(7): 4743–4749.
80. Eggers M. Infectious disease management and control with povidone iodine. *Infect Dis Ther* 2019; 8(4): 581–593.
81. Anderson DE, Sivalingam V, Kang AEZ, et al. Povidone-iodine demonstrates rapid in vitro virucidal activity against SARS-CoV-2, the virus causing COVID-19 disease. *Infect Dis Ther* 2020; 9; 669–675.
82. Matsuyama A, Okura H, Hashimoto S, et al. A prospective, randomized, open-label trial of early versus late povidone-iodine gargling in patients with COVID-19. *Sci Rep* 222; 12: 20449.
83. Pelletier JS, Tessema B, Frank S, et al. Efficacy of povidone-iodine nasal and oral antiseptic preparations against severe acute respiratory syndrome-coronavirus 2 (SARS-CoV-2). *Ear Nose Throat J* 2021; 100(2_suppl): 192S–196S.
84. Arefin MK. Povidone iodine (PVP-I) oro-nasal spray: an effective shield for COVID-19 protection for health care worker (HCW), for all. *Indian J Otolaryngol Head Neck Surg* 2022; 74(Suppl 2): 2906–2911.
85. Zarabanda D, Vukkadala N, Phillips KM, et al. The effect of povidone-iodine nasal spray on nasopharyngeal SARS-CoV-2 viral load: a randomized control trial. *Laryngoscope* 2022; 132(11): 2089–2095.